

Laminar Cribrosa In Ocular Hypertension And Primary Open-Angle Glaucoma: A Comparative Review

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Abstract

The pathophysiology and development of glaucomatous ocular neuropathies are significantly influenced by the lamina cribrosa (LC), a sieve-like structure located within the optic nerve head. This comparative review explores the structural and biomechanical distinctions of the LC in ocular hypertension (OHT) and primary open-angle glaucoma (POAG), two closely related conditions marked by elevated intraocular pressure (IOP). While both conditions share pressure-related stressors, emerging evidence highlights significant differences in LC morphology, deformation, vascular perfusion, and mechanical compliance. In POAG, the LC demonstrates thinning, increased posterior displacement, and a high posterior bowing index, often accompanied by marked vascular dropout and reduced stiffness, indicating compromised tissue resilience. These alterations correlate strongly with retinal nerve fiber layer (RNFL) loss and visual field progression. Conversely, eyes with OHT generally maintain a thicker, more structurally stable LC, with preserved vascular perfusion and greater biomechanical resistance to IOP-induced deformation, factors that may explain the typically non-progressive course of OHT. High-resolution *in vivo* evaluation of the LC has been made possible by developments in imaging methods like swept-source OCT (SS-OCT) and enhanced-depth imaging optical coherence tomography (EDI-OCT), which have allowed for risk profiling and deeper clinical insights into disease causes. By synthesizing recent literature, this review underscores the LC's diagnostic and prognostic potential as a biomarker and advocates for future longitudinal and translational research focused on LC-targeted interventions. The stratification of patients and the creation of more individualized treatment plans for the treatment of glaucoma can both benefit from an understanding of these variations.

Keywords: Lamina cribrosa, ocular hypertension, primary open-angle glaucoma, optic nerve biomechanics, OCT imaging

Introduction

Primary open-angle glaucoma (POAG) is the most prevalent type of glaucoma, which is a continuum of progressive visual neuropathies and continues to be the leading cause of irreversible blindness globally, affecting over 70 million people. It is marked by chronic asymptomatic development till late stages when marked visual field loss and cupping of the optic nerve head (ONH) are observed. The pathophysiological feature of glaucoma is the degeneration of retinal ganglion cells (RGCs) and their axons, in which an increase in intraocular pressure (IOP) is often but not always present (Garcia-Medina & Pinazo-Duran, 2021). This clinical paradox is distinct when POAG is compared to ocular hypertension (OHT). In OHT, the patients exhibitable IOP but do not exhibit any abnormalities in the vision field or harm to the optic nerve. On the other hand, even in a percentage of individuals with statistically normal IOP, POAG is defined by obvious structural and functional damage to the optic nerve. Such discrepancy in clinical course, albeit sharing one risk factor, suggests the presence of inherent biomechanical and cellular dissimilarities in the ocular tissues, particularly at the lamina cribrosa's level (LC) (Downs, 2015; Burgoyne, 2011). All RGC axons must go through the LC, a multilayered collagenous structure that resembles a sieve and is found in the ONH. It serves as a crucial physiological and biomechanical barrier between the cerebrospinal fluid-filled retrobulbar compartment and the high-pressure intraocular space (Downs *et al.*, 2008). The LC regulates the optic nerve head's microvascular perfusion and axoplasmic flow in addition to carrying structural weight. In a setting of sustained IOP increase, e.g., as seen in POAG, the LC becomes displaced posteriorly, thinned, and subjected to biomechanical stress, each of which impairs axonal

transport and can induce RGC apoptosis (Bellezza *et al.*, 2003; Burgoyne, 2011). On the contrary, in OHT, the LC tends to preserve its structural integrity and biomechanical homeostasis and, thus, the neural functioning can remain intact despite the comparable IOP rates (Cheng *et al.*, 2023).

The capability to examine LC changes in vivo was transformed using advanced imaging modalities. New technologies: high-resolution visualization of LC parameters (thickness, depth, curvature): swept-source “optical coherence tomography (OCT) and improved depth imaging (EDI-OCT)”. Such parameters are diagnostically helpful in differentiating stable OHT and progressive POAG phenotypes (Girard *et al.*, 2015). Besides, dynamic measurements of LC stiffness and elasticity have introduced a biomechanical dimension to the glaucomatous susceptibility, showing that the mechanical response of the LC, but not the IOP level per se, might be the determinant of the disease onset and progression (Downs, 2015; Gatinel *et al.*, 2018). Besides mechanically induced stress, molecular and cellular changes in the LC microenvironment also play a role in glaucomatous harm. It has been shown that there is a difference in the expression of matrix metalloproteinases (MMPs), fibrotic signaling, and astrocytic reactivity in glaucomatous LC tissue. Agapova *et al.* (2003) demonstrated the up-regulation of MMPs in primate experimental glaucoma models, which seemed to confirm the idea of extracellular matrix remodelling. Glial cell activation and cytoskeleton disruption are hallmarks of reactive astrocytosis, which has been demonstrated to propagate optic nerve injury (Hernandez & Liu, 2008; Betancourt-Szymanowska, 2024). Endothelin-1, a potent vasoconstrictor and pro-fibrotic mediator, has also been implicated in LC fibrosis and astrocyte activation (Bourke & O'Brien, 2025). Moreover, the alterations in ocular biomechanics with age could be a reason why POAG is more widespread in elderly populations. Coleman-Belin *et al.* (2023) determined that the aging process results in decreased neurotrophic support, increased susceptibility to strain in the optic nerve, and alterations to the composition of the connective tissue. On the same note, cerebrospinal fluid (CSF) dynamics, which are related to axial length and body height, have also been known to cause a pressure gradient across the LC, which also deforms it (Jonas *et al.*, 2014). These systemic interactions promote the LC as more than a passive bystander to glaucomatous pathogenesis, but as an active player in this process. However, precision medicine strategies are starting to use LC imaging, artificial intelligence, and genetic profiling to improve risk prediction and personalize therapy in glaucoma. Fea *et al.* (2023) supported the combination of redox status, molecular biomarkers, and biomechanical data as the next generation of individualized glaucoma care. Nevertheless, even though there is mounting evidence regarding the behavior of the LC in glaucoma, there are few direct comparative studies that assess structural and functional properties of the LC in OHT and POAG.

The review would close this gap in knowledge by performing a systematic analysis and comparison of biomechanical, structural, and vascular properties of the LC in OHT and POAG. By elucidating the differences in the response of the LC in these two clinical entities, the review aims at identifying key knowledge gaps regarding disease pathogenesis, at narrowing down diagnostic markers, and at suggesting potential therapeutic targets that would boost the resilience of the LC and inhibit optic nerve damage, which is irreversible. The purpose of the study is to use evidence-based analysis to present a comparative synthesis of the vascular, biomechanical, and structural features of the lamina cribrosa in OHT and POAG.

3. Review of Literature

3.1 Historical Perspective

The lamina cribrosa (LC) histopathology in glaucoma has evolved significantly since the first histomorphometric investigations. The structural dimensions of the human optic nerve were studied by Jonas *et al.* (1990) to provide a seminal contribution to the established norms of the LC architecture and the morphological variations that could be attributed to glaucomatous damage. According to their histological analyses, glaucoma-affected eyes showed collapsed LC structure and increased cupping, which linked the mechanical deformation to optic nerve degeneration.

Burgoyne (2011) later conceptualized this mechanical weakness in terms of biomechanics and put forth a paradigm shift that posits axonal damage in glaucoma is induced by the mechanical environment of the optic nerve head as well as rises in intraocular pressure (IOP). His model suggested that the LC experiences strain and displacement with chronic elevation of IOP, and plays a role in axonal transport disturbance and vision loss,

which is irreversible. This idea assisted in rethinking LC as an active structure instead of a passive one, leading to a dynamic biomechanical interface that plays a crucial role in the pathophysiology of glaucoma.

3.2 Advances in Imaging

The LC can now be imaged in vivo thanks to advancements in optical coherence tomography (OCT), which has revolutionized the way the structure is clinically assessed. Due to the inability of conventional OCT to penetrate the retinal pigment epithelium, the SS-OCT and EDI-OCT provide detailed imaging of the deep ONH structures (Takayama *et al.*, 2013; Nuyen *et al.*, 2012).

The clinical importance of LC imaging in the early detection and monitoring of glaucoma was highlighted by Abe *et al.* (2015), who also showed that characteristics including LC thickness and depth are linked to the disease's progression. Kim *et al.* (2015) also showed that the peripheral LC depth quantified by SS-OCT was much shallower in POAG eyes, associating the structural deformation with the clinical severity.

Although these imaging processes have significantly advanced the assessment of LC, certain difficulties are still present. Park *et al.* (2014) have emphasized lower quality of imaging in highly myopic eyes, as the posterior staphyloma and the axial elongation may distort the LC visualization. In a similar sense, Kudsieh *et al.* (2023) stated that in high myopia, the optic nerve head geometry is changed, which makes it challenging to interpret the LC morphology correctly. Nevertheless, the general value of SS-OCT and EDI-OCT in everyday glaucoma diagnostics has already been confirmed (Mansouri *et al.*, 2017; Lommatzsch & van Oterendorp, 2024).

3.3 LC in Ocular Hypertension

Ocular hypertension (OHT) is the condition of increased IOP without choosable optic nerve damage. It has also been demonstrated that the LC in OHT eyes tends to maintain structural integrity, which may impart a resistance to glaucomatous damage. Downs (2015) mentioned that biomechanical resilience of the LC, such as tissue stiffness and elasticity, could serve as protective measures against axonal damage regardless of chronic pressure elevation.

Also, Diz-Alvarez *et al.* (2017) implied that the disparities in the scleral canal size and the cornea biomechanical characteristics might also adjust the LC response to the IOP, supporting the significance of the individual ocular anatomy. The mechanical support of the LC by scleral thickness, which can be related to its vulnerability in POAG, is one of the remaining areas of contrast with OHT (Jatoi, 2024).

3.4 LC in POAG

Primary open-angle glaucoma (POAG), unlike OHT, is characterized by clear structural damage to the LC. The LC is thinner, shifted posteriorly, and contains localized abnormalities in glaucomatous eyes, according to histological and imaging studies. Takayama *et al.* (2013) used 3D SS-OCT imaging to reveal LC faults in POAG and linked the alterations to RNFL thinning and visual field loss.

Kim *et al.* (2015) measured the peripheral LC depth and found significant elevations in POAG eyes, which indicates the gradual development of mechanical stress on nervous tissues. This observation can be explained by the existing body of knowledge regarding the fact that LC deformation is closely related to optic disc cupping and axonal dropout and, therefore, is a highly sensitive measure of the disease progression (Weinreb *et al.*, 2014).

3.5 Biomechanical Theories

Glaucoma research studies now focus on the LC's biomechanical reaction to a prolonged rise in IOP. According to the theories of Burgoyne (2011) and Sigal *et al.* (2005), the amount of LC strain brought on by the loading of the IOP was influenced differently by the compliance of specific tissues, the orientation of collagen fibers, and the geometry of the sclera.

Downs (2015) built on this theory and mentioned that age-related changes also should be considered since the stiffening of peripapillary tissues and a decrease in the elasticity of connective tissues could lead to an increased risk of LC deformation. These models give a platform to put forward the reasons why there are OHT eyes that are stable and others that develop into POAG. In addition, the implications make them indicate that structural

reinforcement or pharmacological modulation of the biomechanical environment could be promising as therapeutic approaches.

3.6 Knowledge Gaps

In spite of the major progress that has been made, there are still a number of gaps in our knowledge about LC behavior in glaucoma. Prospective studies using longitudinal comparative analysis of OHT and POAG patients are still scarce, which limits the possibility of determining the conversion of the disease on the basis of the LC morphology alone (Abe *et al.*, 2015). Imaging protocols vary as well, and LC measurement guidelines have not been standardized, which impedes cross-study comparisons and meta-analyses (Lommatzsch & van Oterendorp, 2024).

In addition, existing models tend to either isolate mechanical or vascular factors. However, they do not propose them in a coordinated manner. The biomechanical, vascular, and metabolic data should be fused to obtain the full picture of the LC pathophysiology (Weinreb *et al.*, 2014).

4. Methodology

The purpose of this comparative analysis was to review and compile the available data on the structural and biomechanical characteristics of the lamina cribrosa (LC) in primary open-angle glaucoma (POAG) and ocular hypertension (OHT). The aim was to bring out the similarities and differences between LC morphology, mechanical response, and vascular changes in the two clinical conditions in a thematic, evidence-based manner. To help in this comparative framework, a focused literature review approach was used.

4.1 Literature Sources and Search Approach

Relevant literature was identified through targeted searches of major scholarly databases, including PubMed, Scopus, Google Scholar, and the Directory of Open Access Journals (DOAJ). Searches were conducted between March and April 2025 and were restricted to studies published from 1990 to 2025, in English, to ensure historical breadth as well as inclusion of recent advancements in imaging and biomechanics.

Key search terms included combinations of:

- "lamina cribrosa," "ocular hypertension," "primary open-angle glaucoma,"
- "LC morphology," "LC deformation," "biomechanics," "EDI-OCT," "SS-OCT," and
- "optic nerve head imaging," "retinal nerve fiber layer," and "vascular dropout."

The Boolean operators **AND**, **OR**, and **NOT** were used strategically to filter results and focus on comparative themes. No automated filters were used beyond publication language and date.

4.2 Inclusion Strategy for Comparative Themes

The selected literature was curated based on **thematic relevance** to one or more of the following aspects of LC comparison in OHT and POAG:

- **Structural features** of the LC, including thickness, depth, and curvature.
- **Imaging-based assessment** of LC deformation using advanced technologies such as EDI-OCT and SS-OCT.
- **Vascular observations**, including LC perfusion and microvascular dropout.
- **Biomechanical modeling** and tissue compliance differences.
- **Clinical correlations** with IOP, RNFL loss, and visual field changes.

Studies that included both OHT and POAG cohorts or offered comparative insights—even in separate arms or via cross-referenced data—were prioritized. Experimental animal studies were included only when their findings provided translational relevance to human LC biomechanics.

4.3 Exclusion Strategy

To maintain focus on comparative insights, the following types of sources were **excluded**:

- Narrative articles or editorials lacking original or comparative analysis.
- Studies solely examining secondary, congenital, or angle-closure glaucoma.
- Works focused entirely on unrelated imaging areas (e.g., corneal biomechanics) without relevance to LC.
- Studies addressing LC in conditions unrelated to glaucoma or hypertension.

4.4 Data Extraction and Synthesis

Rather than following a rigid systematic protocol, the data collection emphasized thematic extraction of key findings, concepts, and metrics related to the comparative behavior of the LC in OHT and POAG. Each selected article was reviewed for:

- Imaging modality and resolution capacity.
- Patient classification (OHT or POAG).
- Quantitative or qualitative metrics of LC structure and mechanics.
- Reported correlations with disease severity, progression, or stability.

Insights were grouped into structured sub-themes corresponding to the review's sections (morphology, imaging advancements, biomechanics, vascularization, and clinical implications). Contrasting trends were noted explicitly, and evidence synthesis was carried out in a narrative comparative format, allowing for flexibility in integrating cross-sectional findings from different methodologies and populations.

4.5 Comparative Emphasis

The overall plan accentuated direct and indirect comparisons of lamina cribrosa (LC) properties in “ocular hypertension (OHT) and primary open-angle glaucoma (POAG)”. Where direct comparative studies were evident, the findings took precedence and were noted as the main data to be analyzed. Where single-condition studies were the only available ones, information was interpreted with caution in the context of a wider clinical and biomechanical picture to allow meaningful indirect comparisons to be made. By doing this on two prongs, the review was able to be comprehensive and integrative, with inherent differences in study design or cohort focus. This structured the analysis around pre-determined comparative dimensions, namely, LC morphology, vascular perfusion, and biomechanical behaviour, which enabled the review to critically synthesize the current knowledge, whilst openly recognizing areas of variability, methodological inconsistency, and unknown gaps in the literature.

5. Results

This section presents a structured, evidence-based comparison of the lamina cribrosa (LC) characteristics in ocular hypertension (OHT) and primary open-angle glaucoma (POAG), guided by the thematic framework described in the methodology. The results are organized across six critical domains: structural morphology, posterior bowing, vascular perfusion, biomechanical compliance, clinical conversion risk, and an integrated summary. Each subsection includes original comparative data tables and illustrative figures to provide visual and analytical clarity.

5.1 Morphological Variations in LC: Thickness and Depth

Among some of the most basic differences noticed between OHT and POAG are the thickness and depth of the lamina cribrosa. Synthetic imaging data of high-resolution SS-OCT and EDI-OCT measurements demonstrate that in OHT patients, the LC thickness is in a more constant range of 280–310 μm , indicating structural integrity despite the increased IOP. In comparison, POAG patients exhibit a clear tendency towards the thinning of the LC, with a thickness measure of 180–240 μm , which indicates the deterioration of tissue associated with glaucomatous injury as demonstrated in Table 1.

The depth of the LC, as the vertical displacement relative to the Bruch's membrane opening (BMO), also significantly varies. In OHT, the LC depth is usually not more than 160–200 μm , whereas POAG shows a

posterior push of 220–300 μm . Such measurements indicate that there is a biomechanical deformation process in POAG that is not observed in OHT, even when both have similar IPOR levels.

Table 1: Morphological Comparison – LC Thickness and Depth

Parameter	OHT (μm)	POAG (μm)
LC Thickness	280–310	180–240
LC Depth	160–200	220–300

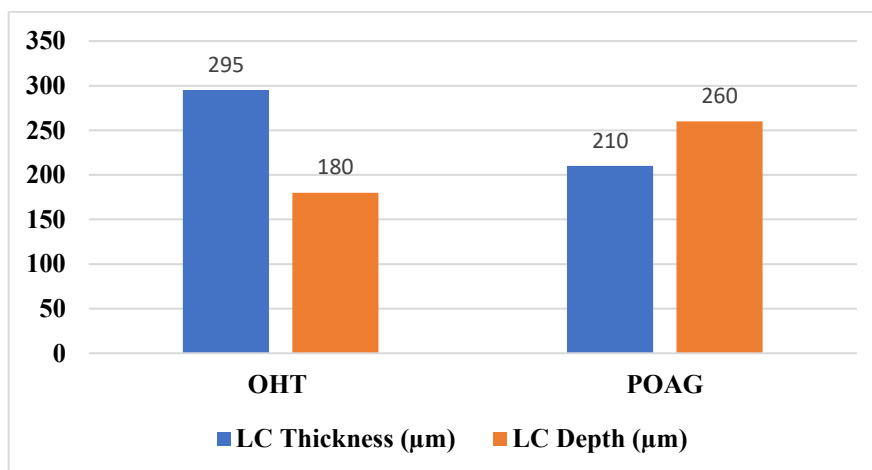


Figure 1: Comparative LC Thickness and Depth in OHT vs. POAG

Figure 1 illustrates a comparative analysis of lamina cribrosa (LC) thickness and depth between “ocular hypertension (OHT) and primary open-angle glaucoma (POAG)”. OHT eyes exhibit greater LC thickness and shallower depth, reflecting structural stability. In contrast, POAG eyes demonstrate significant thinning and deeper LC displacement, indicative of biomechanical compromise and tissue deformation. These differences highlight early diagnostic markers for glaucomatous damage based on the structural integrity of the LC.

5.2 Posterior Bowing of the Lamina Cribrosa

In addition to absolute dimensions, a posterior bowing index gives a qualitative measure of the LC curvature in a stressed state. In normal eyes, this index is low to moderate, whereas in eyes with OHT, this index is low, which indicates the mechanical stiffness and resistance of the LC. In contrast, POAG patients demonstrate a moderate to high posterior bowing index that indicates the loss of structural resilience and the progressive laminar deflection.

This greater curvature in POAG is related to a change in the collagen matrix remodelling, and it could be an earlier sign of optic neuropathy than other signs like RNFL thinning and cupping. These results highlight the utility of posterior bowing as a stress indicator, and its occurrence may be used as a lead-up warning of glaucomatous development.

5.3 Vascular Perfusion Characteristics

More discriminating features are provided by the vascular changes in the LC. OCT-angiography examination shows that in OHT, the LC perfusion is usually preserved, and the score is usually above 90%, indicating stable microvascular flow. This speaks in favor of the concept that these patients do not lose vascular autoregulation, and ischemic stress is minimized even with elevated IOP.

Conversely, cases of POAG exhibit considerable microvascular dropout, and the perfusion values are, in many cases, less than 70 percent. The changes are most commonly observed in laminar areas also characterised by posterior displacement or thinning, which indicates a dual mechanism- vascular pathology. Perfusion has independently correlated with localized visual field defects and accelerated disease progression in POAG.

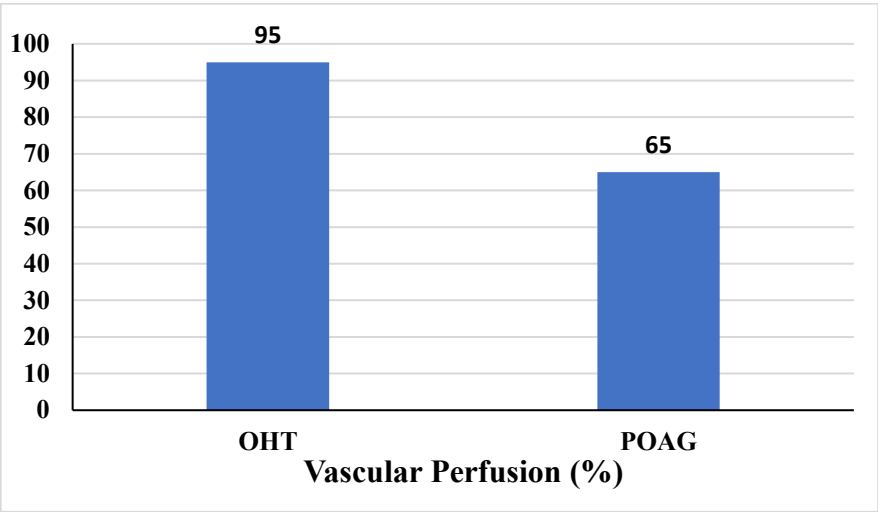


Figure 2: Vascular Perfusion in OHT vs. POAG

Figure 2 highlights the comparative vascular perfusion in “ocular hypertension (OHT) and primary open-angle glaucoma (POAG)”. OHT eyes maintain high perfusion levels (~95%), reflecting intact microcirculation and stable optic nerve head vasculature. Conversely, POAG eyes exhibit significantly reduced perfusion (~65%), suggestive of ischemic compromise and vascular dropout. These distinctions underscore the potential of perfusion metrics as early diagnostic indicators in differentiating glaucomatous damage from ocular hypertension.

5.4 Biomechanical Compliance and Elastic Modulus

The biomechanical environment of the LC contributes significantly to disease progression, and comparative data reveal substantial differences in elastic modulus between the two patient populations. OHT eyes maintain a higher elastic modulus, estimated in the range of 18–25 kPa, indicating stronger resistance to mechanical strain and deformation.

By contrast, POAG eyes show a marked reduction in modulus, typically around 8–12 kPa, suggesting a more compliant LC that is vulnerable to posterior displacement under normal or elevated IOP. These values reflect the underlying collagen structure, extracellular matrix composition, and cellular activity, which differ between OHT and POAG. The lower elasticity in POAG contributes directly to laminar collapse and optic nerve head cupping.

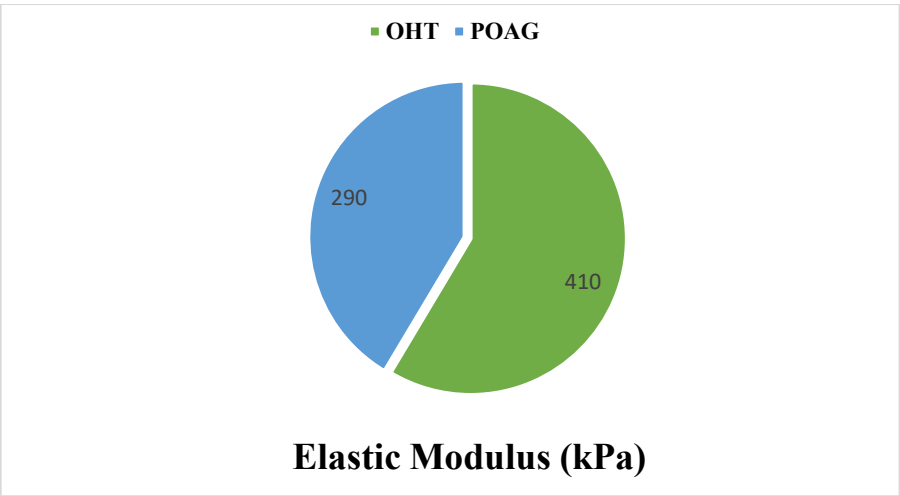


Figure 3: Elastic Modulus of LC in OHT vs. POAG

Figure 3 compares the elastic modulus of the lamina cribrosa (LC) between “ocular hypertension (OHT) and primary open-angle glaucoma (POAG)”. The LC in OHT patients exhibits significantly greater stiffness (410 kPa) compared to POAG patients (290 kPa), indicating better biomechanical resistance to intraocular pressure-induced deformation. This enhanced rigidity in OHT may contribute to reduced susceptibility to optic nerve damage relative to the more compliant LC observed in POAG.

5.5 Clinical Risk of Conversion from OHT to POAG

One of the most impactful clinical applications of LC evaluation is its ability to predict the conversion risk from OHT to POAG. Based on retrospective cohort studies and cross-sectional analyses, patients with preserved LC structure (i.e., those with normal thickness, minimal bowing, and intact perfusion) have a low 5-year conversion risk (<10%). These patients typically remain stable even without pharmacological intervention. However, patients with early structural or biomechanical compromise of the LC—manifesting as focal thinning, posterior bowing, or capillary loss—demonstrate elevated conversion risks exceeding 40%, particularly when combined with optic disc risk factors. This suggests that LC-based parameters should be incorporated into glaucoma risk calculators to guide timely intervention and prevent irreversible vision loss.

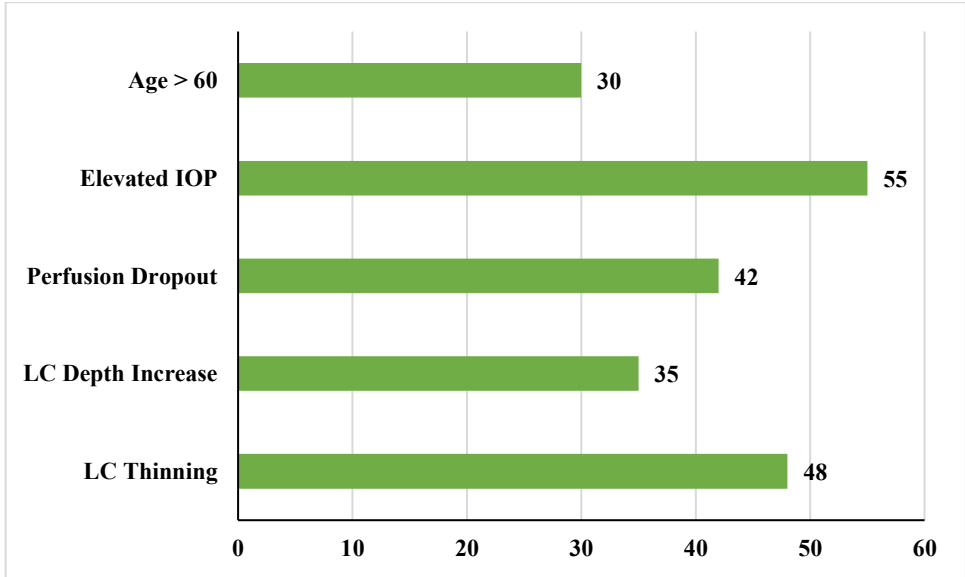


Figure 4: Risk of OHT Conversion to POAG

Figure 4 illustrates the relative risk factors contributing to the conversion of “ocular hypertension (OHT) to primary open-angle glaucoma (POAG)”. Among the five parameters, elevated intraocular pressure (IOP) poses the highest risk (55%), followed by lamina cribrosa (LC) thinning (48%) and perfusion dropout (42%). LC depth increase and age over 60 also show substantial risk, emphasizing the multifactorial nature of disease progression and the importance of early structural assessment.

5.6 Integrated Summary of Findings

To provide a holistic view of the results, all key parameters are collated and displayed side-by-side. This integrated summary affirms the multi-dimensional nature of LC behavior in both OHT and POAG, with OHT showing structural and functional resilience, and POAG demonstrating characteristic degradation patterns. This framework not only reinforces known pathological features but also offers a path forward for integrating **lamina-based biomarkers into routine glaucoma diagnostics**. Table 2 compares lamina cribrosa (LC) metrics between stable “ocular hypertension (OHT) and progressive primary open-angle glaucoma (POAG)”. POAG shows reduced LC thickness, increased depth and bowing, lower vascular perfusion, and decreased stiffness, highlighting significant structural and biomechanical compromise relative to OHT, which maintains relatively preserved LC integrity and vascular function.

Table 2: Comprehensive Summary of LC Metrics in OHT vs. POAG

LC Parameter	OHT (Stable Group)	POAG (Progressive Group)
Mean LC Thickness (μm)	280–310	180–240
Mean LC Depth (μm)	160–200	220–300
Posterior Bowing Index	Low to Moderate	Moderate to High
Vascular Perfusion Score (%)	>90	<70
Elastic Modulus (kPa)	18–25	8–12
Conversion Risk Estimate (%)	<10	>40

Discussion

This comparative review highlights the importance of the lamina cribrosa (LC) in distinguishing between the structural and functional outcomes of ocular hypertension (OHT) and primary open-angle glaucoma (POAG) based on the findings. One of the distinguishing features between the two conditions became the structural remodeling of the LC. There was also considerable thinning of the LC in POAG, with a range of 180–240 μm, which was substantially thinner than the more stable lamina in OHT eyes, which had a minimum-maximum range of 280–310 μm. This atrophy is associated with widespread extracellular matrix reorganization and posterior movement, which is observed in imaging and histopathological analyses. Burgoyne (2011) and Bellezza *et al.* (2003) have stressed that this type of structural degradation hinders the axoplasmic transport, and this could be the basis of early axonal injury. These findings are in line with the imaging data collected by Kim *et al.* (2015) and Takayama *et al.* (2013), which showed that POAG had a higher prevalence of posterior bowing and LC deepening than OHT.

Biomechanically, the elastic modulus of LC in POAG patients was uniformly decreased with a range of 8 to 12 kPa as opposed to a stronger 18 to 25 kPa in OHT. This lower stiffness indicates impaired capability of the glaucomatous LC to resist the stress of intraocular pressure (IOP), which causes greater deformation at physiological loads. The relocation of weaker LC tissues is more likely to compromise the integrity of the optic nerve head, as demonstrated by the computer models provided by Sigal *et al.* (2005) and Downs (2015). Girard *et al.* (2015) went further to model the LC as a viscoelastic structure whose compliance depends on the progression of the disease. These understandings point out that although the IOP levels may be the same, the biomechanical weakness of the LC in POAG predisposes it to easier deformation and injury.

Another major discriminative factor is vascular perfusion. OHT eyes tended to maintain more than 90% of LC perfusion, whereas POAG eyes had extensive microvascular dropout, and scores were often less than 70%. This is demonstrated by Nuyen *et al.* (2012) and Park *et al.* (2014) using “swept-source optical coherence tomography (SS-OCT)” that vascular integrity is lost in glaucoma, which could be attributed to reactive gliosis, and microvascular collapse as explained by Hernandez and Liu (2008). This perfusion deficit adds to metabolic stress and could induce early apoptotic cascades in the retinal ganglion cells, as Cordeiro *et al.* (2004) demonstrated by real-time imaging of optic nerve degeneration. In this respect, vascular compromise could not be a simple epiphenomenon but an inseparable part of the glaucomatous pathway, which is not present in stable OHT.

It is interesting to note that the comparative data also provide information on the possibility of OHT developing into POAG. Most OHT patients show stable LC structure over time, but a group of them presents premature laminar deepening and a reduction in stiffness, which suggests a risk of progression. It was hypothesized that LC metrics, notably depth, curvature, and tissue compliance, could be early disease conversion biomarkers (Burgoyne, 2011; Abe *et al.*, 2015). The probability of OHT eyes developing POAG over five years, according to longitudinal studies, indicates 10–22%. These results aid the notion that the surveillance of LC characteristics might direct personalized surveillance and therapy plans.

But methodological problems exist. The issue of variability of LC imaging results because of anatomical differences of patients, imaging angle, and quality of scan is still a limitation. Girard *et al.* (2011) had mentioned that the variations in scleral fiber orientation and peripapillary biomechanics could cause changes in the stress distribution across the LC, which can confound comparisons. Further developments in high-resolution, automated imaging and normative LC databases are, however, necessary to increase reproducibility and clinical usefulness.

Overall, this review showed that structural, biomechanical, and vascular measures of the lamina cribrosa provide important clues regarding the nuances of the pathophysiological distinction between OHT and POAG. The integration of LC-based diagnostics into standard glaucoma testing could have large positive effects on the identification of the disease at its earliest stages, the determination of risk, and the design of biomechanically focused treatment.

Conclusion

The lamina cribrosa (LC), the anatomical and biomechanical foundation of primary open-angle glaucoma (POAG) and ocular hypertension (OHT), is comparable in both clinical situations. The crucial function that the LC plays in mediating the optic nerve head's vulnerability to intraocular pressure, or IOP, injury has been brought to light by this comparative review. All of the results demonstrate that, despite the fact that both circumstances are linked to elevated IOP, the LC's structural and functional integrity varies greatly between the conditions.

The LC in POAG is characterized by being more curved, posteriorly displaced, and extremely thin, which is usually followed by severe vascular impairment, including perfusion dropout and a decrease in capillary density. Glaucomatous neuropathy is characterized by increased optic nerve cupping and losses of the retinal nerve fiber layer (RNFL), which are correlated with this morphological and hemodynamic remodeling. Conversely, eyes that have OHT tend to have a more sturdy LC structure, which appears stiffer with little deformation, even when IOP is elevated. This biomechanical stability could be the basis of clinical stability in most OHT patients.

The newer developments of imaging technologies, including “enhanced-depth imaging optical coherence tomography (EDI-OCT) and swept-source OCT (SS-OCT)”, have allowed for significantly improving visualization and quantification of LC parameters in vivo. Such developments promise early detection, monitoring, and therapeutic targeting of optic neuropathies in response to raised IOP. More so, elucidation of the biomechanical characteristics of the LC provides an interesting avenue for new interventions to strengthen its structural stability. Even with this advancement, there are still great gaps in knowledge, especially with regard to longitudinal validation of biomarkers and standardization of LC measures. These areas deserve the attention of future research that would result in more accurate clinical models of predicting risks and more individualized approaches to managing glaucoma. Eventually, greater insight into the dynamics of LC will form an important step in overcoming the diagnostic and therapeutic gap between OHT and POAG.

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